# Tracheostomy Tube Failure in Obstructive Sleep Apnea

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*In our institutions we routinely do posttracheostomy sleep studies on patients being treated for obstructive sleep apnea. We have identified several patients who failed to show objective evidence of improvement after tracheostomy. From our studies we have found that both mechanical obstruction and concomitant respiratory control dysfunction caused this failure. A unique tracheostomy tube was constructed to treat the subset of patients with internal collapse of the tracheostomy tube.*

(Rodman DM, Martin RJ: Tracheostomy tube failure in obstructive sleep apnea. West J Med 1987 Jul; 147:41-43)

Once it has been determined that a patient suffers from obstructive sleep apnea, a treatment plan must be formulated. Treatment options include pharmacologic, mechanical and surgical procedures.[1-5] When feasible, follow-up sleep studies are done after administering treatment in an attempt to document success as measured by improved arterial oxygenation and the absence of apneic periods. Tracheostomy has been found to be very successful and, because of this, a subsequent sleep study is often omitted after a surgical procedure.[6,7] We have found that tracheostomy is not uniformly successful and that a follow-up nocturnal oxygen saturation study or formal sleep study may aid in identifying these patients.

We present three cases wherein tracheostomy failed to prevent nocturnal desaturation and apneic periods. The first is a unique case of internal kinking of the tracheostomy tube resulting in persistent obstructive sleep apnea. The second and third cases illustrate the more widely recognized causes of tracheostomy failure in patients with obstructive sleep apnea: external obstruction of the tracheostomy orifice and concurrent central sleep apnea or respiratory drive dysfunction. A discussion of the approach to diagnosis and treatment of tracheostomy failure follows.

## Reports of Cases

### *Case 1*

The patient, a 45-year-old man, presented for evaluation of increasing pedal edema. He had had a 40-kg weight gain to 148 kg (326 lb—280% of ideal body weight) during the year before admission. Subsequently typical symptoms and signs of sleep apnea and right heart failure developed. On physical examination the findings of right heart failure and a short, thick neck confirmed the presence of the obstructive sleep apnea syndrome. Arterial blood gas determinations with the patient breathing room air showed a pH of 7.46, partial carbon dioxide pressure ($Pco_2$) of 36 torr and partial oxygen pressure ($Po_2$) of 62 torr (normal for Denver, pH 7.35 to 7.45, $Pco_2$ 34 to 38 torr and $Po_2$ 65 to 75 torr). Pulmonary function studies showed a forced expiratory volume in one second ($FEV_1$) of 1.3 liters (55% of predicted) and a forced vital capacity (FVC) of 1.75 liters (60% of predicted).

A sleep study was done showing virtually continuous periods of partial and complete obstructive apneas. These were associated with arterial oxygen desaturation to 50%. A regimen of nasal continuous positive airway pressure (CPAP) and supplemental oxygen failed to relieve the apneic periods. A tracheostomy was done.

After the tracheostomy, a sleep study was carried out showing persistent apneic periods associated with arterial oxygen desaturation. A t-piece was used to deliver oxygen, thus eliminating the possibility of obstruction of the tracheostomy orifice, but without benefit. Various lengths and types of plastic endotracheal tubes were used, also without benefit. Using a fiber-optic laryngoscope, the tracheostomy tube was entered. The patient's neck was then actively flexed and extended. When the patient's head was flexed forward, in a position similar to his sleeping position, internal kinking of the tube with complete obstruction was observed.

A noncollapsible endotracheal tube was constructed (Figure 1) and inserted. A follow-up sleep study documented pronounced improvement in nocturnal oxygen saturation with no periods of apnea.

### *Case 2*

The patient, a 37-year-old man, began snoring in his teen years after a weight gain from 73 to 95 kg (161 to 209 lb). His weight then increased to 175 kg (386 lb—310% of ideal body weight). Typical symptoms and signs of sleep apnea developed, with headache, daytime hypersomnolence, restless sleep and hypertension. On laboratory evaluation he had an erythrocytosis, and blood gas determinations with the patient breathing room air showed pH 7.42, $Pco_2$ 34 torr and $Po_2$ 59 torr. Pulmonary function tests showed an $FEV_1$ of 2.85 liters (77% of predicted) and an FVC of 3.68 liters (80% of predicted).

A polysomnographic study showed 30 to 40 partial and complete obstructive apneic periods per hour. Baseline arterial oxygen saturation was 92%, falling to 50% to 70% during apneic periods. Therapeutic trials of nasal CPAP, supplemental oxygen, protriptyline hydrochloride and weight loss were unsuccessful. A tracheostomy was done.

Subsequent to the tracheostomy, his symptoms persisted.

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ABBREVIATIONS USED IN TEXT

CPAP = continuous positive airway pressure
$FEV_1$ = forced expiratory volume in 1 second
FVC = forced vital capacity
$Pco_2$ = partial carbon dioxide pressure
$Po_2$ = partial oxygen pressure

The results of a repeat sleep study were unchanged from baseline. During sleep, the external tracheostomy orifice was observed to be obstructed by the patient's soft tissues. Protecting the orifice with a tracheostomy collar failed to ameliorate the problem. A neck support (Figure 2) was constructed, after which a sleep study showed resolution of the apneic periods. The patient has tolerated the prosthesis well, with total resolution of symptoms.

*Case 3*

The patient, a 69-year-old man, was admitted for evaluation of mental status changes and hypercapnia. He had long-standing chronic bronchitis and for several years had typical symptoms and signs of sleep apnea. On presentation he was a somnolent, obese man with physical findings of right heart failure. Arterial blood gas determinations with the patient breathing two liters of nasal oxygen showed a pH of 7.28, $Pco_2$ 75 torr and $Po_2$ 48 torr. After vigorous therapy with steroids and bronchodilators, his blood gas values improved to pH 7.39, $Pco_2$ 66 torr and $Po_2$ 55 torr. Pulmonary function tests showed an FVC of 1.63 (38% of predicted) and $FEV_1$ of 1.21 (42% of predicted). Thyroid function was normal. A sleep study was done showing 75 partial and complete episodes of apnea per hour and 21 episodes of hypopnea per hour—associated with decreased but present respiratory effort. These periods were associated with arterial oxygen saturation as low as 28%. A tracheostomy was done.

A sleep study carried out 14 days after the tracheostomy showed a decreased number of apneas but persistent periods of hypopnea with arterial oxygen desaturation to 80%. Arterial blood gases measured with the patient breathing 35% oxygen showed pH 7.36, $Pco_2$ 74 torr and $Po_2$ 57 torr. A regimen of progesterone, 20 mg taken orally every eight hours, was started. A follow-up sleep study after seven days of therapy showed elimination of the hypopneic periods and maintenance of arterial oxygen saturation at greater than 90%. A follow-up arterial blood gas study with the patient breathing room air was pH 7.42, $Pco_2$ 46 torr and $Po_2$ 61 torr.

## Discussion

*Clinical Approach to Diagnosis and Treatment*

In each of our patients, a posttracheostomy sleep study showed a lack of improvement in nocturnal oxygenation and continued periods of disordered breathing. As these cases show, there are several possible reasons for this failure.

The first is external obstruction of the tracheostomy orifice due to redundant submandibular and neck soft tissue. This diagnosis can often be made by observing the patients during sleep. They will be noted to flex their heads forward, obstructing the orifice. If this is noted, treatment of the disorder can often be effected by protecting the tracheostomy orifice with a tracheostomy collar. If this proves ineffective, a supportive device (Figure 2) can be constructed to maintain the head in a neutral position during sleep.

If these measures prove ineffective, then a second cause, internal obstruction of the tracheostomy tube, should be suspected. This again may be due to redundant neck and submandibular soft tissue. In this case the tracheostomy tube itself kinks or is obstructed at the distal end. In addition to redundant soft tissue, distal obstruction may be due to tracheal stenosis or granuloma formation occurring months to years after a tracheostomy. Internal obstruction can be differentiated from external obstruction by administering oxygen directly with a t-piece while documenting persistent periods of obstructive apnea and arterial desaturation. Direct observation with a fiber-optic laryngoscope is useful to confirm that a properly sized tracheostomy tube is in place and to document the internal obstruction.

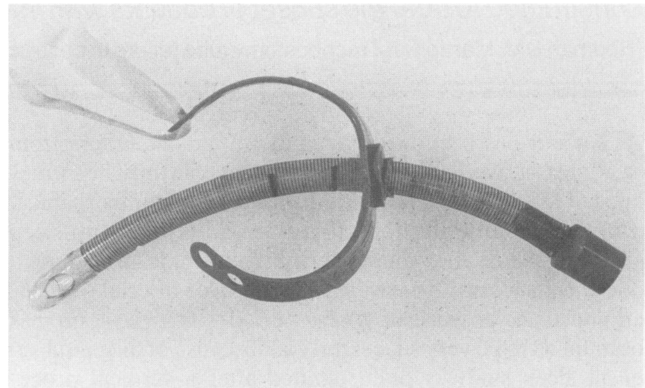

**Figure 1.**—A tracheostomy tube was constructed with a wire coil-reinforced endotracheal tube.

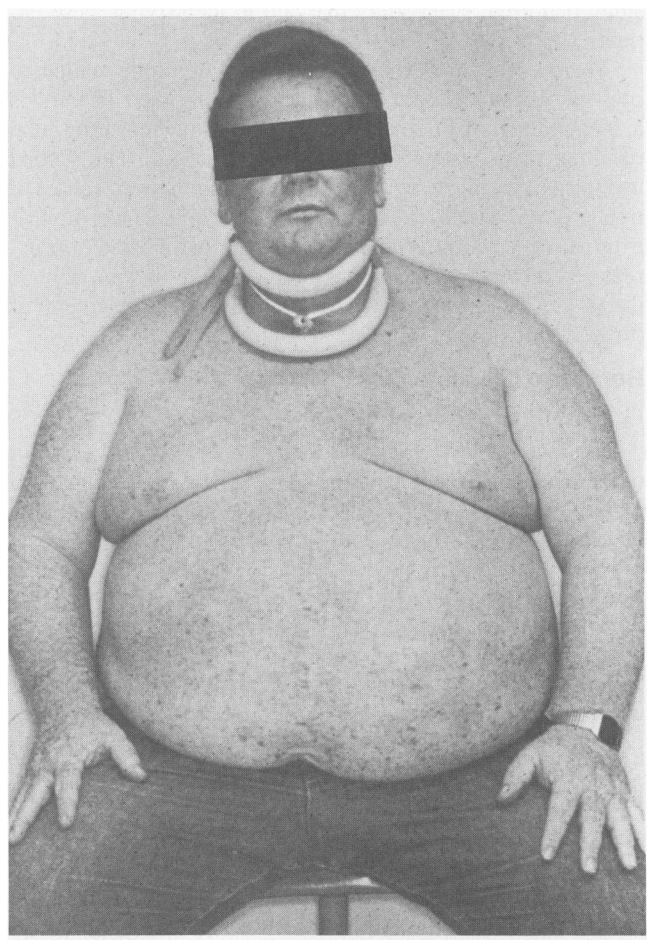

**Figure 2.**—The photograph shows patient 2 with supportive device in place.

We have found that all commercially available types of plastic tracheostomy tubes are ineffective in these cases. Metal tracheostomy tubes, while likely to be effective, are often not well tolerated by patients. For these reasons, we have constructed a tracheostomy tube using a coiled wire-reinforced type of tube (Figure 1).* The tube length is determined by direct measurement using a fiber-optic laryngoscope. The tube is then fitted with a standard 15-mm adapter and flanges.

If these mechanical measures fail to correct the nocturnal desaturation, then concurrent central sleep apnea or respiratory drive dysfunction should be suspected. It is rare for these disorders to go undiagnosed on a pretracheostomy sleep study. Their importance relative to the obstructive component, however, may be difficult to determine before a tracheostomy.[8] A repeat sleep study will show persistent hypopnea or central apneic periods if these are the cause. Because of the possibility of improvement in central drive after a tracheostomy, this study should be done several weeks after the procedure. Paradoxically, patients with mixed-type apneas (both decreased effort and upper airway obstruction) may, in some cases, show a slight increase in the number of central apneas and hypopneas following tracheostomy.[7] Blood gases done with the patient breathing room air may show resting hypercapnia out of proportion to the degree of lung disease if respiratory drive dysfunction is the cause. This can be confirmed by documenting an appropriate fall in the arterial $P_{CO_2}$ during coached hyperventilation or by formal respiratory drive studies. If these causes are shown, then treatment with either respiratory stimulants or diaphragmatic pacing should be instituted.[3,9,10]

*The authors thank Shirley Phister, RN, for expert advice on constructing tracheostomy tubes.

A final cause of nocturnal arterial oxygen desaturation following tracheostomy should be noted. Exacerbations of ventilation perfusion mismatch due to left ventricular failure and chronic obstructive lung disease may occur at night. In addition, accumulation of airway secretions may worsen at night. The respiratory pattern may be normal or show periodic hypopneas or a Cheyne-Stokes pattern.[11,12] Therapy in these cases should be directed at the underlying problem and may include the use of supplemental oxygen.

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